



Original research | Оригинальное исследование  
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# Prediction of adverse outcomes in the long-term follow-up period in patients with chronic heart failure who have suffered a myocardial infarction

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## Abstract

**Aim** – to determine the prognostic significance of global longitudinal strain of the left ventricle (GLS) and soluble stimulating growth factor (sST2) in patients with chronic heart failure (CHF) after myocardial infarction (MI) in the annual follow-up period.

**Material and methods.** The study included 96 patients with CHF who were hospitalized with acute MI. All subjects underwent speckle-tracking echocardiography and determination of concentrations of sST2, vascular endothelial growth factor (VEGF), N-terminal pro-brain natriuretic peptide (NT-proBNP) and C-reactive protein (CRP). After 12 months patients were assessed for cases of stroke, recurrent myocardial infarction, hospitalization for unstable angina or decompensation of CHF, and cardiovascular death, forming a combined endpoint (CEP).

**Results.** The development of CEP was registered in 44 (45.8%) patients with initially lower left ventricular ejection fraction and GLS, higher left ventricular myocardial mass index, index of impaired local contractility, basal

diameter of the excretory tract, as well as a higher score on the Syntax scale and concentrations of CRP, NT-proBNP and sST2. During the ROC-analysis for the development of CEP, optimal thresholds for sST2 and NT proBNP were determined, which were 36.1 ng/ml and 427 pg/ml, respectively. The multifactorial analysis made it possible to develop a mathematical model for predicting adverse outcomes within 12 months after MI, which included such indicators as GLS – odds ratio (OR) 0.51 (0.39; 0.72), the number of points on the Syntax scale – OR 3.05 (2.2; 6.8), concentrations of NTproBNP – OR 2.9 (1.45; 5.1) and sST2 – OR 3.3 (1.65; 7.51).

**Conclusion.** The developed prognostic model includes factors reflecting various links in the pathogenesis of CHF, which provides an integrated approach to assessing the risks of recurrent cardiovascular events after MI.

**Keywords:** chronic heart failure, myocardial infarction, global longitudinal strain (GLS), soluble stimulating growth factor (sST2), prognostic model.

**Conflict of interest:** nothing to disclose.

## Citation

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# Прогнозирование неблагоприятных исходов в отдаленном периоде наблюдения у пациентов с хронической сердечной недостаточностью, перенесших инфаркт миокарда

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## Аннотация

**Цель** – определить прогностическую значимость глобальной продольной деформации левого желудочка (GLS) и растворимого рецептора стимулирующего фактора роста (sST2) у пациентов с хронической сердечной недостаточностью (ХСН), перенесших инфаркт миокарда (ИМ), в годовом периоде наблюдения.

**Материал и методы.** В исследование включено 96 пациентов с ХСН, госпитализированных с острым ИМ. Всем обследуемым проведена спекл-

трекинг эхокардиография и определение концентраций sST2, фактора роста эндотелия сосудов (VEGF), N-концевого предшественника мозгового натрийуретического пептида (NT-проBNP) и C-реактивного белка (СРБ). Через 12 месяцев оценены случаи развития острого нарушения мозгового кровообращения, повторного ИМ, госпитализаций по поводу нестабильной стенокардии или декомпенсации ХСН и сердечно-сосудистой смерти, составивших комбинированную конечную точку (ККТ).

**Результаты.** Развитие ККТ зарегистрировано у 44 (45,8%) пациентов, имевших исходно более низкие показатели фракции выброса левого желудочка (ЛЖ) и GLS, более высокие показатели индекса массы миокарда ЛЖ, индекса нарушения локальной сократимости, базального диаметра выводного тракта, а также более высокий балл по шкале Syntax и концентраций СРБ, NT-proBNP и sST2. При проведении ROC-анализа в отношении развития комбинированной конечной точки были определены оптимальные пороговые значения для sST2 и NTproBNP, составивших 36,1 нг/мл и 427 пг/мл соответственно. Проведенный многофакторный анализ позволил разработать математическую модель прогнозирования неблагоприятных исходов в течение 12 месяцев после перенесенного ИМ,

в которую вошли такие показатели, как величина GLS – отношение рисков (ОР) 0,51 (0,39; 0,72), количество баллов по шкале Syntax – ОР 3,05 (2,2; 6,8), концентрации NTproBNP – 2,9 (1,45; 5,1) и sST2 – ОР 3,3 (1,65; 7,51).

**Выводы.** Разработанная прогностическая модель включает факторы, отражающие различные звенья патогенеза ХСН, что обеспечивает комплексный подход к оценке рисков развития повторных сердечно-сосудистых событий после перенесенного ИМ.

**Ключевые слова:** хроническая сердечная недостаточность, инфаркт миокарда, глобальная продольная сократимость, растворимый рецептор стимулирующего фактора роста, прогностическая модель.

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#### Список сокращений

ХСН – хроническая сердечная недостаточность; СН – сердечная недостаточность; ФВ ЛЖ – фракция выброса левого желудочка; ИБС – ишемическая болезнь сердца; ИМ – инфаркт миокарда; АГ – артериальная гипертензия; БМ – биологический маркер; КАГ – коронароангиография; СД – сахарный диабет; ФП – фибрилляция предсердий; АКШ – аортокоронарное шунтирование; БАБ – бета-адреноблокатор; иАПФ – ингибитор ангиотензинпревращающего фермента; АМР – агонист минералокортикоидных рецепторов; БКК – блокатор кальциевых каналов; ЛКА – левая коронарная артерия; ПНА – передняя нисходящая артерия; ОА – огибающая артерия; ПКА – правая коронарная артерия; ЧКВ – чрескожное коронарное вмешательство; ОНМК – острое нарушение мозгового кровообращения; ПИКС – постинфарктный кардиосклероз; ДААТ – двойная антиагрегантная терапия; ЛДФ – лазерная доплеровская флоуметрия; ККТ – комбинированная конечная точка; НС – нестабильная стенокардия; ОШ – отношение шансов; ДИ – доверительный интервал; ИММ – индекс массы миокарда; ИНЛС – индекс нарушения локальной сократимости; ШОКС – шкала оценки клинического состояния; ВД – выводной тракт; КДР – конечный диастолический размер; КСР – конечный систолический размер; РКК – резерв капиллярного кровотока; ЛНП – липопротеиды низкой плотности.

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## ■ INTRODUCTION

Over the past twenty years, chronic heart failure (CHF) has acquired the character of an “epidemic”. CHF is widespread throughout the world and affects more than 60 million people, and, according to experts, the number of people with this pathology will increase [1]. Despite the impressive body of scientific data on the pathophysiology of heart failure (HF) and the possibilities of surgical and drug treatment and prevention, this pathology is associated with significant morbidity and mortality, especially among older people [2–3].

Left ventricular ejection fraction (LVEF) is a fundamental factor for risk stratification in patients with coronary artery disease (CAD), in particular after myocardial infarction (MI). However, being within the normal range, it does not allow a full recognition of the degree of deformation changes in the myocardium. Today, there is compelling evidence for the prognostic value of LV global longitudinal strain (GLS), which is more valuable than LVEF, in stratifying the risks of adverse clinical outcomes in different groups of patients with cardiovascular diseases. Although GLS is an independent predictor of death, left ventricular remodeling and hospitalization of HF patients with aortic stenosis, acute MI, congestive HF, this indicator is not used in clinical practice widely enough [4–6].

Numerous studies on post-infarction remodeling of the myocardium confirm the close connection between the biological markers (BM) of endothelial dysfunction, fibrosis and inflammation, and development and progression of HF [7–8]. At the same time, despite the processes underlying heart failure being widely known, the studies of prognostic value of biomarkers showing the undergoing structural and functional changes of the

cardiovascular system, particularly in the development of MI, with respect to the progression of adverse cardiovascular events both in the short-term and long-term perspective remain a priority in modern cardiology [9].

The most commonly used B-type natriuretic peptides in clinical practice, in particular the N-terminal precursor of brain natriuretic peptide (NT-proBNP), are very useful in diagnosis, risk stratification and determination of optimal treatment; however, they have well-known limitations, since their level is dependent on such factors as renal dysfunction, age, obesity, atrial fibrillation, and some other cardiologic and non-cardiologic diseases [10].

The ST2 growth-stimulating factor is a member of the superfamily of interleukin-1 (IL-1) receptors that exists in two forms: transmembrane receptor (ST2L) and the soluble receptor (sST2). The natural ligand of the ST2 is the IL-33. Due to a complex action that IL-33 causes to tissue damage and inflammation, it is involved in the pathogenesis of some diseases (e.g., allergy, autoimmune diseases, cancer, atherosclerosis, and diabetes). It is most important that IL-33 plays a cardio-protective role preventing fibrosis and hypertrophy of the myocardium as a response to mechanical load with the help of ST2L. The damage of the myocardium or mechanical stress stimulate release of the sST2 that competes with ST2L for binding the IL-33, thus inhibiting the positive effects caused by the interaction of ST2L/IL-33, so, the excess of sST2 may assist development of myocardial fibrosis and ventricular remodeling [11].

It was proven that the sST2 provides important information about the prognosis in heart failure (both acute and chronic), and it is less affected by the renal function, age, body mass index, and disease etiology

than the natriuretic peptides. Although the sST2 is not yet widely used, it can be easily measured multiple times in emergencies and daily clinical practice [11].

## AIM

To determine the prognostic significance of GLS and sST2 in patients with CHF after myocardial infarction in the annual follow-up period.

## MATERIAL AND METHODS

The study was carried out in the Cardiology Departments No. 1 and No. 2 of the Clinics of the Samara State Medical University in the period from 2021 to 2022. In included 96 patients with CHF urgently hospitalized for in-patient treatment in the acute period of MI not more than 24 hours after the event.

**Inclusion criteria:** age over 18; signing of voluntary informed consent for participation in the study; previous diagnosis of 'chronic heart failure' of I, II and III functional classes; established diagnosis of 'myocardial infarction' at the moment of signing of the informed consent; availability of coronary angiography (CAG).

**Exclusion criteria:** decompensated diabetes mellitus; confirmed autoimmune diseases; aggravated oncological history; decompensated renal or hepatic failure; diseases of the blood system; history of coronary artery bypass grafting (CABG); other factors of myocardium geometry change (refractory hypertension, hemodynamically significant congenital and acquired cardiac defects, dilatation and ischemic cardiomyopathy).

Myocardial infarction and CHF were diagnosed in compliance with the effective clinical recommendations [12–14].

The clinical and anamnestic characteristics of patients are shown in Table 1. Among the participants included in the study, male patients prevailed, and the median age was 64.5 (57.0; 72.3) years. In the vast majority of patients, there was a history of arterial hypertension (AH), and CHF of NYHA II functional class. History of previous MI was known in 25 (26%) patients, and that of acute cerebrovascular event, in 7 (7.3%). The diagnosed diabetes mellitus was present in 18 (18.8%) individuals. In most cases (61.6%), nob Q-wave myocardial infarction was found, the incidence rate of the anterior localization of MI was 46.9%. Signs of acute heart failure as per Killip II-III were found in 14.6% patients. In 10 patients, atrial fibrillation (AT) rhythm was found, in eight of those, as a complication of the infarction in progress. The median score of patients with CHF on the Rating scale of clinical state (RSCS) was 5 (3; 6).

In-patient treatment was also compliant with the current clinical recommendations [12–14]. In accordance therewith, the medication therapy as of the moment of patients' inclusion in the study contained antiaggregants, anticoagulants (as needed), beta-blockers (BB), statins, inhibitors of angiotensin converting enzyme (ACEI) or sartans, mineralocorticoid receptor agonists (AMR), loop diuretics, calcium channel blockers (CBB) (Table 1). All patients with AF received oral anticoagulants.

Characteristics	Abs. Value	%
<b>Sex</b>		
female	31	32.3
male	65	67.7
PICS	25	26
Smoking	3	3.1
AH	90	93.8
DM	18	18.8
ACVA	7	7.3
AF	10	10.4
<b>CHF, functional class (NYHA)</b>		
II	88	91.7
III	8	8.3
<b>Severity of AHF (Killip)</b>		
I	82	85.4
II	8	8.3
III	6	6.3
<b>IM type</b>		
without Q wave	59	61.5
with Q wave	37	38.5
<b>IM localization</b>		
anterior LV wall	45	46.9
posterior LV wall	51	53.1
<b>Prescribed therapy</b>		
DAAT	94	97.9
Statins	93	96.9
ACEI/sartans	87	90.6
BB	90	93.8
AMR	70	72.9
Loop diuretics	52	54.2
CBB	33	34.4
<b>Results of angiographic investigation</b>		
PCI performed at the hospital	61	63.5
<b>Surgical tactics on discharge</b>		
Indicated for CABG	24	25
Indicated for second-stage PCI	34	35.4
Conservative therapy	38	39.6

Notes: PICS: post-infarction cardiosclerosis; DAAT: double anti-aggregate therapy.

**Table 1.** Clinical and anamnestic characteristics of the patients included in the study (n=96)

**Таблица 1.** Клинико-анамнестическая характеристика пациентов, включенных в исследование (n=96)

Based on CAG data, the current condition of the coronary bed was assessed: lesions of the trunk of the left coronary artery (LCA), left anterior descending artery (ADA), circumflex artery (CX), and right coronary artery (RCA). Afterwards, the severity of coronary bed lesion was evaluated using the Syntax scale. In the event a stenosis over 70% was found, the lesion of the coronary artery was deemed significant. In cases of RCA and trunk of LCA, stenosis over 50% was considered hemodynamically significant. Considering the clinical and angiographic picture, and with the patient's consent, the surgical tactics and time frame of intervention were identified (CABG and/or stenting of the symptom-dependent artery).

Following the results of CAG, in 58 (60.4%) patients triple-vessel disease of the coronary bed was found, in 24 (25%) patients, there was found hemodynamically significant lesion of two coronary arteries, in 20 (20.8%), lesion of the LCA trunk. Percutaneous coronary intervention (PCI) was performed in more than 60% cases. Further surgical tactics (CABG) was identified for 24

Factor	No CEP, n=52	Onset of CEP, n=44	OR (95% CI)	P
Median age, years	61.5 (56.5; 72)	66 (59.5; 73)	1.02 (0.98; 1.07)	0.257
Male sex, n (%)	35 (67.3)	30 (68.2)	1.04 (0.44; 2.46)	0.927
DM, n (%)	10 (19.2)	8 (18.2)	1.08 (0.4; 2.95)	0.881
AF, n (%)	7 (13.5)	3 (6.8)	0.47 (0.11; 1.94)	0.288
History of ACVA, n (%)	3 (5.8)	4 (9.1)	1.63 (0.35; 7.73)	0.533
History of MI, n (%)	12 (23.1)	13 (29.5)	1.4 (0.56; 3.49)	0.472
AH, n (%)	50 (96.2)	40 (90.9)	0.4 (0.07; 2.3)	0.29
MI with Q-wave, n (%)	17 (32.7)	20 (45.5)	1.72 (0.74; 3.93)	0.2
Anterior MI, n (%)	24 (46.2)	21 (47.7)	1.07 (0.48; 2.38)	0.878
Killip II–III, n (%)	5 (9.6)	9 (20.5)	2.42 (0.75; 7.85)	0.134
PCI during hospitalization, n (%)	34 (65.4)	27 (61.4)	0.84 (0.37; 1.94)	0.683

**Table 2.** Clinical and anamnestic factors influencing the development of adverse cardiovascular events within 1 year

**Таблица 2.** Клинико-анамнестические факторы, влияющие на развитие неблагоприятных сердечно-сосудистых событий в течение 1 года

(25%) patients; second-stage PCI for 34 (35.4%) patients; conservative therapy, for 38 (39.6%) patients (**Table 1**).

On the third day of hospitalization, patients' venous blood was sampled to identify concentrations of such biomarkers as sST2 ("Presage ST2 Assay" test system, "Critical Diagnostics", USA), vascular endothelial growth factor (VEGF) ("Human VEGF-A ELISA Kit, Bender MedSystems GmbH", Austria) and NTproBNP ("NT-proBNP" test system, "Biomedica", Austria). The assessments of the total and biochemical blood count, as well as blood coagulation, lipid, and CRP tests were performed within the routine clinical practice.

The ECG was registered on the "CardiovitAT 2" apparatus ("Schiller", Switzerland) in twelve standard leads.

Echocardiography (EchoCG) and 2D mode EchoCG speckle tracking were performed on the "Philips EPIQ 5" ultrasound machine (USA) in accordance with the Guidelines of the American Society of Echocardiography and the European Association for Cardiovascular Imaging [15].

During the hospital period, all patients underwent laser Doppler flowmetry (LDF) in order to determine microcirculation disorders using the general practitioner's laser blood microcirculation analyzer "LAKK-OP" ("Lazma" LLC, Russia).

In the course of the first phase of the study, no cases of hospital death were registered. In the second phase, telephone inquiry was used to assess the disease progress and clinical outcomes within 12 months. The combined end point (CEP) in this study is represented by cases of mortality due to cardiovascular pathology and recurrent cardiovascular events: ACVA, new incidences of IM, unstable angina (UA), and hospitalization for in-patient treatment of CHF decompensation.

**Statistical processing of data** was performed in the statistical calculation software suite R 4.3.2 (R Foundation

for Statistical Computing, Austria). Descriptive statistics are represented as absolute and relative frequencies for qualitative variables, and medians (Me) with interquartile range (Q1; Q3) for quantitative variables. Intergroup differences for quantitative variables were assessed using the Mann–Whitney test between two independent samples; for categorical variables, the Fisher's exact test. The determination of prognostic factors for the occurrence of recurrent events was performed using univariate regression analysis with calculation of the odds ratio (OR) and determination of its 95% confidence interval (CI). The prediction of the study criteria was also assessed by performing ROC (receiver operating characteristics) curve analysis, in which the area under the curve (AUC) values were calculated.

## RESULTS

Within the 12 months of observation, the onset of CEP was registered in 44 (45.8%) participants of the study: cardiovascular death took place in 2 (2.1%) cases, 8 (8.3%) patients were hospitalized due to UA and the same number, due to development of recurrent IM, 24 (25%) people were hospitalized due to CHF decompensation, and 2 (2.1%), due to development of ACVA. Based on that, groups of patients were identified with favorable and unfavorable progress in the long term-post-infarction period.

The assessment of effect of clinical and anamnestic characteristics on the probability of development of recurrent cardiovascular events within 12 months from the moment of patients' inclusion in the study revealed no statistically significant associations with sex, age, MI localization, AHF severity as per Killip scale, PCI in the period of hospitalization, history of MI, DM, AH, ACVA, and the presence of AF and NYHA functional class CHF ( $p > 0.05$  in all cases) (**Table 2**).

At the same time, the patients with an adverse clinical outcome in the year-long follow-up, a statistically significant higher score of RSCS was registered, (5 (4; 7) vs. 4 (3; 5) in patients with a favorable progress of post-MI period,  $p < 0.001$ ). The regression analysis revealed that high RSCS scores were significant predictors of development of recurrent cardiovascular events in CHF patients within one year after MI (OR=1.44 (95% CI: 1.17; 1.82),  $p = 0.001$ ).

The comparative analysis of basic EchoCG findings and CAG results (**Table 3**) revealed that in the event of development of adverse cardiovascular events the patients originally had statistically significant lower values of left ventricle ejection fraction (LVEF) and global longitudinal strain (GLS), and statistically significant higher values of end-systolic length (ESL) and end-diastolic length (EDL) of the left ventricle, end-systolic volume of the left ventricle (ESV), myocardial mass index of the left ventricle (LV MMI), wall motion score index (WMSI), basal diameter of the LV outflow tract (LV OT), and higher scores as per Syntax scale. Besides, patients with an adverse long-term outcome demonstrated diastolic dysfunction of the left ventricle (LV DD) significantly more often (70.5%). The groups with favorable and unfavorable clinical prognoses did not differ in the value



Factor	No CEP, n=52	Onset of CEP, n=44	P	OR (95% CI)	p for OR
LV EF*, %	54 (49.5; 57.7)	48 (39; 54)	<b>&lt;0.001</b>	0.9 (0.84; 0.95)	<b>&lt;0.001</b>
EDR*, mm	48 (44.5; 50)	51 (48; 53.5)	<b>0.002</b>	1.11 (1.02; 1.21)	<b>0.018</b>
ESR*, mm	33 (30; 37)	36 (32; 42)	<b>0.004</b>	1.09 (1.02; 1.17)	<b>0.013</b>
ESV*, ml	118.5 (106.5; 139)	129 (114; 151.5)	0.15	1.01 (0.99; 1.02)	0.261
EDV*, ml	55 (45.5; 70.5)	65.5 (56; 86.5)	<b>0.003</b>	1.03 (1.01; 1.05)	<b>0.013</b>
LV OT dia., mm					
proximal*	28.5 (27; 31)	30 (27; 32)	0.416	1.06 (0.95; 1.19)	0.273
distal*	25 (25; 26.5)	25 (24; 26.5)	0.732	0.98 (0.86; 1.11)	0.748
basal*	35 (32; 38)	38 (35; 40)	<b>0.01</b>	1.08 (1; 1.18)	<b>0.045</b>
median*	29.5 (27; 31)	30 (27; 32)	0.744	1.02 (0.92; 1.14)	0.699
GLS*, %	19.8 (18.45; 20.3)	16.65 (15.1; 17.65)	<b>&lt;0.001</b>	0.37 (0.26; 0.54)	<b>&lt;0.001</b>
LV MMI*, g/m2	100 (80.5; 110.35)	107 (97.5; 124)	<b>0.013</b>	1.02 (1; 1.04)	<b>0.018</b>
WMCI*	1.19 (1; 1.4)	1.41 (1.1; 1.8)	<b>0.008</b>	4.6 (1.55; 15.4)	<b>0.009</b>
LA vol.*, ml	62 (53.5; 68)	65.5 (57; 79.5)	0.089	1.02 (0.99; 1.04)	0.202
PPAsyst*, Hgmm	30.5 (27; 35.5)	32.5 (27.5; 40.5)	0.145	1.05 (1; 1.1)	0.073
IV LA*, ml/m2	334 (28; 40)	335 (30.75; 42.33)	0.264	1.0 (0.98; 1.01)	0.526
IV RA*, ml/m2	25.2 (22; 28)	26.6 (22.8; 30.55)	0.197	1.01 (0.96; 1.07)	0.639
Vtr*, m/s	2.39 (2.22; 2.6)	2.53 (2.25; 2.8)	0.094	3.27 (1.09; 9.82)	<b>0.033</b>
LV DD, n (%)	26 (50)	31 (70.5)	<b>0.042</b>	2.39 (1.02; 5.55)	<b>0.042</b>
Syntax*, pts.	15.5 (8.5; 21.25)	38 (31; 41)	<b>&lt;0.001</b>	1.2 (1.13; 1.29)	<b>&lt;0.001</b>
Number of affected coronary arteries					
Single-vessel, n (%)	11 (21.2)	3 (6.8)	0.14	1.58 (0.96; 2.74)	0.081
Two-vessel, n (%)	12 (23.1)	12 (27.3)			
Three-vessel, n (%)	29 (55.8)	29 (65.9)			
LCA trunk disease, n (%)	11 (21.2)	9 (20.5)	0.933	0.96 (0.35; 2.58)	0.933

Notes: \* values are represented as Me (Q1; Q3); IV LA: indexed volume of left atrium; IV RA: indexed volume of right atrium.

**Table 3.** Comparative analysis of echocardiographic parameters and coronary angiography results depending on the development of CEP

**Таблица 3.** Сравнительный анализ эхокардиографических показателей и результатов КАГ в зависимости от развития ККТ

of the end-diastolic volume of LV (EDV), proximal, distal and median diameter of the LV OT, volumetric values of the left atrium (LA) and right atrium (RA), value of systolic pressure in the pulmonary artery (PPAsyst), and the number of affected coronary arteries and presence of LCA trunk lesion. The statistically significant predictors of development of recurrent cardiovascular events were as follows: LV EF (OR (95% CI) – 0.9 (0.84; 0.95),  $p < 0.001$ ), EDR, ESR, ESV, basal diameter of LV OT (OR (95% CI) – 1.08 (1; 1.18),  $p = 0.045$ ), GLS (OR (95% CI) – 0.37 (0.26; 0.54),  $p < 0.001$ ), LV MMI (OR (95% CI) – 1.02 (1; 1.04),  $p = 0.018$ ), WMCi (OR (95% CI) – 4.6 (1.55; 15.4),  $p = 0.009$ ), tricuspid regurgitation velocity (Vtr) (OR (95% CI) – 3.27 (1.09; 9.82),  $p = 0.033$ ), presence of LV DD (OR (95% CI) – 2.39 (1.02; 5.55),  $p = 0.042$ ), and the score on the Syntax scale (OR (95% CI) – 1.2 (1.13; 1.29),  $p < 0.001$ ) (**Table 4**). It is noteworthy that neither the number of affected coronary arteries nor the presence of hemodynamically significant damage to the left coronary artery trunk had a significant impact on the long-term prognosis of patients after MI.

When analyzing the microcirculation parameters obtained during LDF, it was found that patients who developed repeated adverse cardiovascular events during 12 months of follow-up, initially had statistically significantly lower values of the microcirculation index (MI), the amplitudes of blood flow oscillations in the myogenic (Am), neurogenic (An) and endothelial (Ae) frequency ranges, the respiratory test index (RTI) and the Hurst exponent (R/S), as well as statistically significantly higher capillary blood flow reserve (CBFR) (**Table 4**). The groups did not differ in the value of variation coefficient

(Kv), values of relative perfusion saturation of blood flow with oxygen (Sm), specific consumption of oxygen in the tissues (I), relative entropy (H0), and correlation dimensions of phase pattern (D2). The obtained data indicate that patients with an unfavorable prognosis had more manifested disturbances of microvascular hemodynamics during the year, as well as a decrease in the amplitude-frequency spectrum of perfusion oscillations.

The odds of development of recurrent cardiovascular events within one year were statistically significantly associated with values of RTI (OR (95% CI) – 0.81 (0.66; 0.97),  $p = 0.025$ ) and CBFR (OR (95% CI) – 1.15 (1.05; 1.27),  $p = 0.005$ ). Besides, with the Ae value growing by 0.1 the odds ratio of adverse outcome development reached 0.58 (95% CI: 0.37; 0.88), An – 0.32 (95% CI: 0.14; 0.67), Am – 0.52 (95% CI: 0.32; 0.8), R/S – 0.62 (95% CI: 0.39; 0.96) and D2 – 0.68 (95% CI: 0.46; 0.97).

The analysis of levels of general laboratory parameters depending on the development of adverse cardiovascular events within one year after an index MI showed that the patients with adverse outcomes had significantly higher levels of CRP, NTproBNP and sST2, and the calculated glomerular filtration rate (GFR) was significantly lower than the patients with a favorable progression of the remote follow-up period (**Table 5**). The groups did not differ in the levels of troponin, creatine phosphokinase (CPK) and its MB isoform (CPKMB), total cholesterol (TC), low-density lipoproteins (LDL) and VEGF, although there was a tendency towards higher values of glycemia in patients with unfavorable prognosis ( $p = 0.06$ ). The statistically significant predictors of recurrent events were the value of GFR calculated using the CKD-EPI formula

Factor	No CEP, n=52	Onset of CEP, n=44	P	OR (95% CI)	p for OR
MI, pf. u.	15.3 (14.4; 16.3)	14.5 (13.7; 15.9)	<b>0.042</b>	0.76 (0.57; 1)	0.055
Kv, %	6.93 (5.06; 8.51)	6.18 (4.73; 7.81)	0.194	0.89 (0.75; 1.06)	0.205
Ae, pf. u.	0.58 (0.52; 0.64)	0.52 (0.44; 0.61)	<b>0.007</b>	0.58 (0.37; 0.88)	<b>0.013</b>
An, pf. u.	0.57 (0.54; 0.61)	0.54 (0.48; 0.57)	<b>0.002</b>	0.32 (0.14; 0.67)	<b>0.004</b>
Am, pf. u.	0.51 (0.44; 0.55)	0.42 (0.37; 0.51)	<b>0.002</b>	0.52 (0.32; 0.8)	<b>0.004</b>
RTI, %	35.5 (34.2; 37)	34.2 (32.6; 38.7)	<b>0.038</b>	0.81 (0.66; 0.97)	<b>0.025</b>
CBFR, %	127 (124; 129)	130 (125; 133)	<b>0.008</b>	1.15 (1.05; 1.27)	<b>0.005</b>
Sm, c. u.	4.35 (4.03; 4.6)	4.08 (3.87; 4.65)	0.175	0.58 (0.22; 1.46)	0.249
I, c. u.	33.7 (30.8; 36)	32.1 (29.8; 34.7)	0.229	0.95 (0.85; 1.05)	0.282
R/S	0.47 (0.4; 0.52)	0.4 (0.32; 0.48)	<b>0.02</b>	0.62 (0.39; 0.96)	<b>0.036</b>
H0	0.34 (0.31; 0.38)	0.34 (0.3; 0.36)	0.197	0.58 (0.24; 1.39)	0.23
D2	1.43 (1.36; 1.5)	1.39 (1.25; 1.49)	0.063	0.68 (0.46; 0.97)	<b>0.039</b>

Note: the factor values are gives as medians Me (Q1; Q3).

**Table 4.** Comparative analysis of microcirculation parameters depending on the development of CEP

**Таблица 4.** Сравнительный анализ параметров микроциркуляции в зависимости от развития ККТ

(OR (95% CI) – 0.95 (0.92; 0.98),  $p=0.006$ ), glucose levels (OR (95% CI) – 1.17 (1.01; 1.36),  $p=0.029$ ), CRP (OR (95% CI) – 1.06 (1.02; 1.11),  $p=0.004$ ), NT-proBNP (OR (95% CI) – 1.03 (1.02; 1.08),  $p<0.001$ ), and sST2 (OR (95% CI) – 1.13 (1.07; 1.2),  $p<0.001$ ).

Thus, development of adverse cardiovascular events in CHF patients after MI is associated within the following year with decrease of global deformation of the left ventricle, severity of coronary atherosclerosis, disruption of microcirculation, presence of kidney dysfunction, hyperglycemia and elevation of levels of such biomarkers as CRP, NTproBNP and sST2. At the same time, the clinical and anamnestic characteristics as predictors of long-term adverse outcomes become secondary.

To predict the risk of development of recurrent cardiovascular events, a mathematical model was developed with step-by-step selection of predictors that showed their significance in the development of combined endpoint in the single factor analysis, as well as predictors that have wide evidence with respect to riskometry, with exclusions based on the Akaike's information criterion (AIC). The analysis included criteria that showed their prognostic value in a univariate analysis, and the generally accepted risk factors with a vast body of evidence: patient's age and sex, history of DM, history of MI, type of MI and severity of CHF in the Killip class, score on the RSCS and Syntax scales, number of involved coronary arteries, values of LV EF, GLS, LV MMI, RCII, ESL, EDL, ESV, basal diameter of the LV OT and Vtr, presence of LV DD,

RTI and CFBR, Hurst exponent, correlation dimensions of the phase pattern and amplitude-frequency spectrum of perfusion oscillations, as well as concentrations of glucose, CRP, NT-proBNP, sST2, TC and LDL, and CKD-EPI calculated GFR.

Following the results of the analysis, the final model (**Table 6**) included such factors as GLS value, points on the Syntax scale, levels of NTproBNP and sST2. The risk ratio (RR) for NTproBNP was 2.9 (1.45; 5.1), for the Syntax scale, 3.05 (2.2; 6.8), for sST2, 3.3 (1.65; 7.51), and for GLS, 0.51 (0.39; 0.72).

The resulting model was characterized by a Nagelkerke pseudo  $R^2$  value of 0.7 (adjusted value – 0.66), Sommers DXY rank correlation of 0.89 (adjusted value – 0.86) and AUC of 0.94 (95% CI: 0.89–0.97) (adjusted value – 0.93) (**Fig. 1**).

Following the results of the multivariate regression analysis, we derived the regression equation using the following mathematical formula:

$P=1/(1+e^{-(Bo + B1*x1 + B2*x2 + B3*x3 + B4*x4)})$ , where:

$e$  – base of natural logarithm (2.718);  $Bo$  – constant (1.32);  $B_1$  – coefficient for NTproBNP (0.012);  $B_2$  – coefficient for GLS (-0.74);  $B_3$  – coefficient for Syntax scale points (0.13);  $B_4$  – coefficient for sST2 (0.15);  $x_1$  – concentration of NTproBNP, pg/ml;  $x_2$  – GLS value, %;  $x_3$  – number of Syntax scale points;  $x_4$  – concentration of sST2, ng/ml.

Thus, the equation can be written as follows:

$P=1/(1+e^{-(1.32 + 0.012*x1 - 0.74*x2 + 0.13*x3 + 0.15*x4)})$ .

Factor	No CEP, n=52	Onset of CEP, n=44	P	OR (95% CI)	p for OR
Glucose, mmol/l	6.0 (5.3; 7.3)	6.6 (5.6; 8.4)	0.06	1.17 (1.01; 1.36)	<b>0.029</b>
Troponin, pg/ml	708.5 (174; 2283.1)	616.4 (89.8; 3205.5)	0.953	1.0 (1.0; 1.0)	0.146
CPK, U/l	306.9 (120; 778.2)	315.5 (143.5; 796.8)	0.678	1.0 (1.0; 1.0)	0.707
CPKMB, U/l	31.1 (21.3; 70.6)	47.5 (21.3; 77.8)	0.617	0.99 (0.82; 1.0)	0.439
CKD-EPI GFR, ml/min/1.73m <sup>2</sup>	77 (74; 80)	67 (61; 77)	<b>0.003</b>	0.95 (0.92; 0.98)	<b>0.006</b>
TC, mmol/l	5.1 (4.4; 5.65)	5.05 (4.75; 5.4)	0.8	0.86 (0.58; 1.22)	0.396
LDL, mmol/l	3.07 (2.58; 3.9)	3.28 (2.74; 3.32)	0.376	0.94 (0.61; 1.42)	0.759
CRP, mg/l	14.9 (7.9; 21.1)	22.3 (12.5; 31.9)	<b>0.004</b>	1.06 (1.02; 1.11)	<b>0.004</b>
NT-proBNP, pg/ml	192.4 (111.2; 517.8)	1339.7 (605.4; 1886.9)	<b>&lt;0.001</b>	1.03 (1.02; 1.08)	<b>&lt;0.001</b>
sST2, ng/ml	27.2 (21.3; 34.8)	45.8 (37; 63.4)	<b>&lt;0.001</b>	1.13 (1.07; 1.2)	<b>&lt;0.001</b>
VEGF, pg/ml	387 (187.5; 461.6)	249.7 (153.8; 370)	0.116	0.79 (0.53; 1.17)	0.248

Note: the factor values are gives as medians Me (Q1; Q3).

**Table 5.** Levels of the main laboratory parameters depending on the development of CEP

**Таблица 5.** Уровни основных лабораторных показателей в зависимости от развития ККТ

Predictor	$\beta$ (SE)	RR	95% CI	p	VIF
Constant	1.32	-	-	-	-
NTproBNP, ng/ml	0.012	2.9	1.45; 5.1	0.018	1.34
GLS, %	-0.74	0.51	0.39; 0.72	0.032	1.27
Syntax, points	0.13	3.05	2.2; 6.8	0.002	1.05
sST2, ng/ml	0.15	3.3	1.65; 7.51	0.041	1.08

**Table 6.** Coefficients in the model for predicting the risk of recurrent cardiovascular events

**Таблица 6.** Коэффициенты в полученной модели прогнозирования риска развития повторных сердечно-сосудистых событий

To ensure the ease of use of this formula, a calculator for calculating the probability of development of recurrent cardiovascular events in patients with CHF within a year after a previous MI was designed on its basis. After the required parameters are filled in (NTproBNP and sST2 levels, Syntax scale points and GLS value), the program provides the result of probability of development of recurrent cardiovascular events within 12 months in percent (**Fig. 2**).

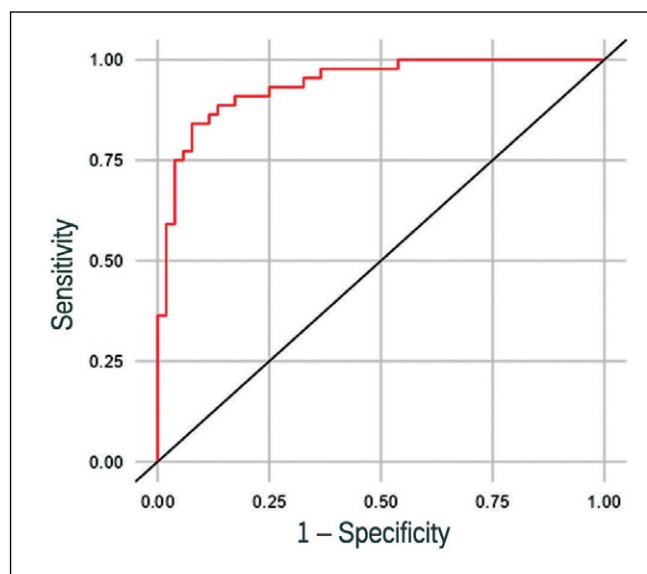
Using a predicted event probability of 60% as a threshold, the resulting model had an accuracy of 87.5% (95% CI: 84.2; 91.3), a sensitivity of 81.8% (95% CI: 72.6; 90.8), and a specificity of 92.3% (95% CI: 87.8; 98.1). The positive predictive value was 92.3% (95% CI: 83.9; 96.4).

## DISCUSSION

Heart failure is a complex clinical syndrome that occurs in various diseases, including MI, characterized by pathological changes in the structure and/or function of the heart [16].

Currently, there are many studies confirming the prognostic significance of various predictors of adverse outcome in patients with HF. These include EchoCG data, results of effort tests, and levels of such biomarkers as NTproBNP, galectin-3, hs-T-troponin, and CRP. At the same time, despite the identification of many prognostic markers, clinical decision-making in CHF is still based primarily on parameters such as the presence of HF symptoms (NYHA class), LVEF, QRS duration and morphology [17].

Although LVEF is an important indicator in the diagnosis and monitoring of patients with CHF, in some cases, LVEF assessment may not be informative enough and may not reflect the severity of the clinical condition, particularly at the onset of HF. Recent studies suggest that myocardial strain is a more sensitive parameter for assessing cardiac function than LVEF. Thus, P. Janwetchasil *et al.* (2024) studied the prognostic value of GLS using the MRI of the heart in patients with known or suspected coronary heart disease with preserved systolic function of the left ventricle. The multivariate analysis showed that patients with GLS less than 14.4% had a significantly higher risk of adverse cardiovascular events compared with patients with GLS greater than 14.4% [6]. L. Caunite *et al.* (2024) showed that GLS improves stratification of risk based on LVEF in patients after MI with elevation of the ST segment (STEMI). The study included 1909 STEMI patients, the average follow-up



**Рисунок 1.** ROC-кривая для предсказаний, полученных с использованием модели.

**Figure 1.** ROC-curve for predictions obtained using the model.

being 69 months. The cumulative 10-year survival rate was 91% in patients with improvement or slight decline in GLS compared with 85% in patients with a GLS decline of >7% within one year after the index event. In a multivariate regression analysis, a decrease in GLS >7% from baseline remained independently associated with the development of the endpoint after adjustment for clinical and echocardiographic parameters of left and right ventricular function. Thus, speckle tracking strain echocardiography has the potential to improve risk stratification in patients with STEMI, even with preserved or moderately reduced LVEF at baseline and in follow-up [4]. The correlation between the GLS value and level of BNP with the development of pathological remodeling after the MI was demonstrated earlier by V.E. Oleinikov *et al.* (2022). Patients with pathological LV remodeling on days 7–9 of MI had statistically significantly lower GLS values, and within 6 months the proportion of patients with low and intermediate LVEF was 24.4% and 60%, respectively. A decrease in GLS of less than 11.7% was a

**Figure 2.** Example of using a calculator for riskometry in patients with CHF who had MI

**Рисунок 2.** Пример использования калькулятора для рискометрии у пациентов с ХСН, перенесших ИМ.

highly sensitive and specific predictor of the development of post-infarction pathological LV dilation [18]. In the present study, a dynamic assessment of the GLS value was not performed, but only the effect of GLS on the development of late adverse cardiovascular events was assessed. At the same time, the prognostic significance of GLS was demonstrated by both univariate and multivariate analyses, which makes this indicator a valuable marker for early risk stratification in patients with CHF who have had an MI, regardless of its dynamics.

Recently, the prognostic significance of ST2 and its advantages over natriuretic peptides gained much attention. The clinical effectiveness of assessing the level of sST2 in HF was confirmed by numerous studies conducted over the past 20 years. A meta-analysis of 2016 studied 7 clinical trials with more than 6372 participants and confirmed the prognostic value of sST2 with respect to adverse outcomes in CHF patients [19]. It is known that an increase in sST2 levels after MI has a long-term prognostic value for the development of CHF. According to clinical observations, patients with elevated sST2 levels after MI were more susceptible to subsequent maladaptive myocardial remodeling and progression of HF [20]. sST2 has also been shown to be a powerful predictor of adverse clinical events in AHF. Xue-Qing Guan et al. (2024) increased the predictive value for risk stratification in patients with AHF over a three-year follow-up period by assessing the incidence of major adverse cardiovascular events, defined as re-hospitalization for HF and/or all-cause mortality. The authors found that the optimum threshold value for sST2 was 34 ng/ml. Patients above this threshold had higher rates of re-hospitalization and mortality, which emphasized the prognostic importance of elevated sST2 levels in the study population. The diagnostic value of sST2 varied across different HF patient groups, particularly in patients with a history of MI, indicating the importance of considering prognostic differences between patient groups when monitoring sST2 levels in clinical practice [21].

To assess the risks of developing adverse outcomes within a year for the studied cohort of patients, we proposed a mathematical model for determining the risk of developing recurrent cardiovascular events and death within 12 months in patients with CHF after MI. The multivariate regression analysis established the following as independent predictors of development of adverse clinical outcomes: GLS value, score on the Syntax scale, and concentration of sST2 and NTproBNP identified not later than two days after the development of MI. It is to be noted that in this study, the NTproBNP did not lose its prognostic value. It was proven that the inclusion of new biomarkers in the multi-parametric models, in addition to the widely known natriuretic peptides, significantly improves the risk stratification, and the highly sensitive troponins and the sST2 seem to be more reliable biomarkers for the stratification of risk [22–23].

## CONCLUSION

The assessment of levels of GLS and sST2 in patients with IM, along with the assessment of the severity of the coronary atherosclerosis and the level of NTproBNP is a promising tool that can improve early stratification of risk and diagnostic and therapeutic management of patients admitted for emergency care. The increase of sST2 levels in plasma is likely associated with significant activation of both neurohormonal and profibrotic mechanisms, which may help identify patients at high risk of early adverse LV remodeling after MI.

The use of the proposed prognostic model in clinical practice, in particular at the hospital stage, will allow not only to stratify the risk of developing recurrent cardiovascular events in patients with CHF after a previous MI and promptly identify individuals with a high probability of developing adverse clinical events within the next year, but also to provide a more personalized approach to treatment and preventive measures for a specific patient. ■

ADDITIONAL INFORMATION	ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ
<b>Study funding.</b> The study was the authors' initiative without external funding.	<b>Источник финансирования.</b> Работа выполнена по инициативе авторов без привлечения финансирования.
<b>Conflict of interest.</b> The authors declare that there are no obvious or potential conflicts of interest associated with the content of this article.	Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с содержанием настоящей статьи.
<b>Compliance with Ethical Standards.</b> The authors confirm that the rights of the people who participated in the study were respected, including obtaining informed consent.	Соответствие нормам этики. Авторы подтверждают, что соблюдены права людей, принимавших участие в исследовании, включая получение информированного согласия.
<b>Contribution of individual authors.</b> Yu.A. Trusov: conducting of the scientific research and experiment; writing of the article. Yu.V. Shchukin: approval of the research concept, scientific consulting, editing of the manuscript. L.V. Limareva: evaluation of laboratory research methods, development of correlation models based on biomarker data in patients with CHF. All authors gave their final approval of the manuscript for submission, and agreed to be accountable for all aspects of the work, implying proper study and resolution of issues related to the accuracy or integrity of any part of the work.	<b>Участие авторов.</b> Ю.А. Трусов – проведение научного исследования и эксперимента; написание текста статьи. Ю.В. Щукин – утверждение концепции исследования, научное консультирование, редактирование рукописи. Л.В. Лимарева – оценка лабораторных методов исследования, разработку корреляционных моделей на основании данных биомаркеров у пациентов с различными фенотипами ХСН. Все авторы одобрили финальную версию статьи перед публикацией, выразили согласие нести ответственность за все аспекты работы, подразумевающую надлежащее изучение и решение вопросов, связанных с точностью или добросовестностью любой части работы.



## REFERENCES / ЛИТЕРАТУРА

1. Groenewegen A, Rutten FH, Mosterd A, Hoes AW. Epidemiology of heart failure. *Eur J Heart Fail.* 2020;22(8):1342-1356. DOI: [10.1002/ehf.1858](#)
2. Ignatieva VI, Kontsevaya AV, Lukyanov MM, et al. Cost-effectiveness analysis of increasing drug therapy coverage for patients with coronary artery disease in combination with atrial fibrillation and heart failure. *Cardiovascular Therapy and Prevention.* 2024;23(4):3950. [Игнатиева В.И., Концевая А.В., Лукьянов М.М., и др. Клинико-экономический анализ увеличения охвата лекарственной терапией пациентов с ишемической болезнью сердца в сочетании с фибрилляцией предсердий и хронической сердечной недостаточностью. *Кардиоваскулярная терапия и профилактика.* 2024;23(4):3950]. DOI: [10.15829/1728-8800-2024-3950](#)
3. Roger VL. Epidemiology of Heart Failure: A Contemporary Perspective. *Circ Res.* 2021;128(10):1421-1434. DOI: [10.1161/CIRCRESAHA.121.318172](#)
4. Caunite L, Myagmardorj R, Galloo X, et al. Prognostic Value of Follow-up Measures of Left Ventricular Global Longitudinal Strain in Patients With ST-Segment Elevation Myocardial Infarction. *J Am Soc Echocardiogr.* 2024;37(7):666-673. DOI: [10.1016/j.echo.2024.03.007](#)
5. D'Ávila LBO, Lima ACGBD, Milani M, et al. Left ventricular global longitudinal strain and cardiorespiratory fitness in patients with heart failure: Systematic review and meta-analysis. *Hellenic J Cardiol.* 2024;79:58-69. DOI: [10.1016/j.hjc.2023.09.010](#)
6. Janwetchasil P, Yindeengam A, Krittayaphong R. Prognostic value of global longitudinal strain in patients with preserved left ventricular systolic function: A cardiac magnetic resonance real-world study. *J Cardiovasc Magn Reson.* 2024;26(2):101057. DOI: [10.1016/j.jcmr.2024.101057](#)
7. Jenkins WS, Roger VL, Jaffe AS, et al. Prognostic Value of Soluble ST2 After Myocardial Infarction: A Community Perspective. *Am J Med.* 2017;130(9):1112.e9-1112.e15. DOI: [10.1016/j.amjmed.2017.02.034](#)
8. Felker GM, Anstrom KJ, Adams KF, et al. Effect of Natriuretic Peptide-Guided Therapy on Hospitalization or Cardiovascular Mortality in High-Risk Patients With Heart Failure and Reduced Ejection Fraction: A Randomized Clinical Trial. *JAMA.* 2017;318(8):713. DOI: [10.1001/jama.2017.10565](#)
9. Khamitova AF, Lakman IA, Akhmetvaleev RR, et al. Multifactor predictive model in patients with myocardial infarction based on modern biomarkers. *Kardiologiya.* 2020;60(3):14-20. [Хамитова А.Ф., Лакман И.А., Ахметвалеев Р.Р., и др. Многофакторная прогностическая модель у пациентов с инфарктом миокарда в отдаленном периоде на основе современных биомаркеров. *Кардиология.* 2020;60(3):14-20]. DOI: [10.18087/cardio.2020.3.2593](#)
10. Clerico A, Emdin M. Diagnostic Accuracy and Prognostic Relevance of the Measurement of Cardiac Natriuretic Peptides: A Review. *Clin Chem.* 2004;50(1):33-50. DOI: [10.1373/clinchem.2003.024760](#)
11. Riccardi M, Myhre PL, Zelniker TA, et al. Soluble ST2 in Heart Failure: A Clinical Role beyond B-Type Natriuretic Peptide. *JCDD.* 2023;10(11):468. DOI: [10.3390/jcdd10110468](#)
12. Barbarash OL, Duplyakov DV, Zateichnikov DA, et al. 2020 Clinical practice guidelines for Acute coronary syndrome without ST segment elevation. *Russian Journal of Cardiology.* 2021;26(4):4449. [Барбараш О.Л., Дупляков Д.В., Затеищikov Д.А., и др. Острый коронарный синдром без подъема сегмента ST электрокардиограммы. Клинические рекомендации 2020. *Российский кардиологический журнал.* 2021;26(4):4449]. DOI: [10.15829/1560-4071-2021-4449](#)
13. Russian society of cardiology. 2020 Clinical practice guidelines for Acute ST-segment elevation myocardial infarction. *Russian Journal of Cardiology.* 2020;25(11):4103. [Российское кардиологическое общество. Острый инфаркт миокарда с подъемом сегмента ST электрокардиограммы. Клинические рекомендации 2020. *Российский кардиологический журнал.* 2020;25(11):4103]. DOI: [10.15829/29/1560-4071-2020-4103](#)
14. Russian society of cardiology. 2020 Clinical practice guidelines for Chronic heart failure. *Russian Journal of Cardiology.* 2020;25(11):4083. [Российское кардиологическое общество (РКО). Хроническая сердечная недостаточность. Клинические рекомендации 2020. *Российский кардиологический журнал.* 2020;25(11):4083. DOI: [10.15829/1560-4071-2020-4083](#)
15. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American society of echocardiography and the European association of cardiovascular imaging. *J Am Soc Echocardiogr.* 2015;28(1):1-39.e14. DOI: [10.1016/j.echo.2014.10.003](#)
16. McDonagh TA, Metra M, et al. 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur J Heart Fail.* 2024;26(1):5-17. DOI: [10.1002/ehf.3024](#)
17. Chuda A, Banach M, Maciejewski M, Bielecka-Dabrowa A. Role of confirmed and potential predictors of an unfavorable outcome in heart failure in everyday clinical practice. *Ir J Med Sci.* 2022;191(1):213-227. DOI: [10.1007/s11845-020-02477-z](#)
18. Oleinikov VE, Golubeva AV, Galinskaya VA, et al. Early markers of pathological left ventricular remodeling in patients after ST-elevation myocardial infarction according to speckle-tracking echocardiography. *Russian Journal of Cardiology.* 2022;27(6):4837. [Олейников В.Э., Голубева А.В., Галимская В.А., Бабина А.В., Донецкая Н.А., и др. Ранние маркеры формирования патологического ремоделирования левого желудочка у больных после инфаркта миокарда с подъемом сегмента ST по результатам speckle tracking эхокардиографии. *Российский кардиологический журнал.* 2022;27(6):4837]. DOI: [10.15829/1560-4071-2022-4837](#)
19. Aimo A, Vergaro G, Passino C, et al. Prognostic Value of Soluble Suppression of Tumorigenicity-2 in Chronic Heart Failure. *JACC Heart Failure.* 2017;5(4):280-286. DOI: [10.1016/j.jchf.2016.09.010](#)
20. Bière L, Garcia G, Guillou S, et al. ST2 as a predictor of late ventricular remodeling after myocardial infarction. *Int J Cardiol.* 2018;259:40-42. DOI: [10.1016/j.ijcard.2018.02.058](#)
21. Guan XQ, Guan L, Cheng G, et al. Examining the Long-Term Prognostic Significance of Serum sST2: Influence of Myocardial Infarction History and Subgroup Level Standardization. *J Inflamm Res.* 2024;17:7733-7744. DOI: [10.2147/JIR.S482475](#)
22. Pascual-Figal DA, Manzano-Fernández S, Boronat M, et al. Soluble ST2, high-sensitivity troponin T- and N-terminal pro-B-type natriuretic peptide: complementary role for risk stratification in acutely decompensated heart failure. *Eur J Heart Fail.* 2011;13(7):718-725. DOI: [10.1093/eurjhf/hfr047](#)
23. Clemente G, Soldano JS, Tuttolomondo A. Heart Failure: Is There an Ideal Biomarker? *Rev Cardiovasc Med.* 2023;24(11):310. DOI: [10.31083/j.rcm2411310](#)